Erratum

In Volume 61, No. 10 (December 1985) the following figure and caption should be substituted for the figure that was used on page 895.

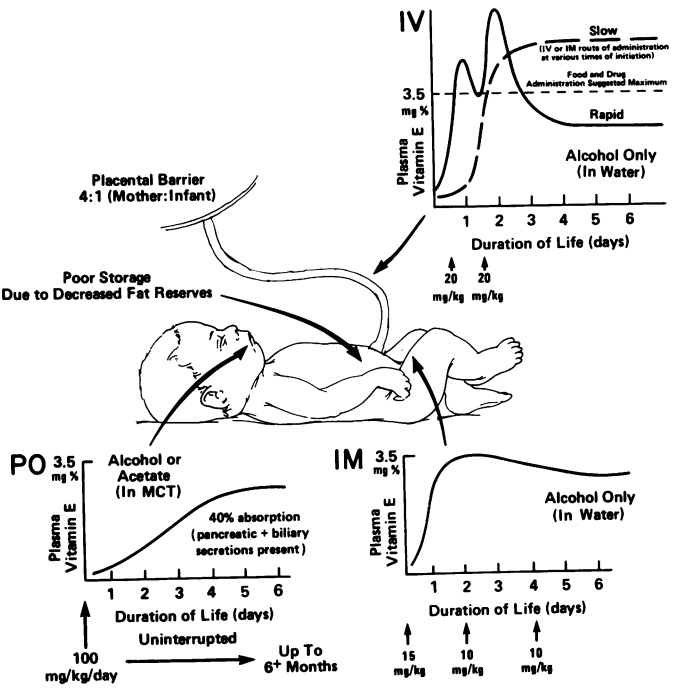

Fig. 9. Schematic diagram showing the vitamin E status of the deficient preterm infant and our current recommendations for vitamin E supplementation based on the spindle cell pathogenesis of retinopathy of prematurity. Graphs summarize the relative relationship between plasma vitamin E (mg%) and days of life for oral, intramuscular and intravenous routes of administration. PO: The slow plasma rise in the first week of life is representative of oral administration of vitamin E in medium chain triglycerides (MCT). This is our recommended route of administration to suppress the development of severe retinopathy of prematurity.[4] IM: The rapid, nonpeaking plasma rise in the first week of life is representative of aqueous, intramuscular administration of vitamin E (alcohol). This is our recommended route of administration to suppress the development of severe intraventricular hemorrhage (IVH) or if the infant must be NPO for longer than three days.[4] Both oral and intramuscular initial routes of administration are equally efficacious in protecting spindle cells from the initial cluster of oxidative insults. IV: The early, high plasma peaks in the first week of life (———) are representative of the levels obtained by rapid intravenous administration of vitamin E in one subset of infants in the Phelps' clinical trial (20 mg/kg on days 1 and 2).[11] These peaks are associated with the toxicities of retinal and intraventricular hemorrhages, especially in infants $\leq$1,000 grams birth weight. The prolonged, high plasma plateau targeted by Johnson by the slow intravenous or intramuscular routes of administration (———) is associated with the toxicities of sepsis and necrotizing enterocolitis.[16] The Food and Drug Administration has warned that levels above 3.5 mg% (----) should be avoided.